

Effects of inert gas narcosis on the vestibular ocular reflex

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Hamilton K, Porlier G, Landolt J, Fraser W, Fowler B. Effects of inert gas narcosis on the vestibular ocular reflex. *Undersea Biomed Res* 1986; 13(3):355-359.—A study was conducted to examine the vestibular ocular reflex (VOR) during narcosis. The slow phase velocity of the nystagmus was measured in six subjects by means of electronystagmography during the inhalation of 25% nitrous oxide. It was found that nitrous oxide increased the velocity of the slow phase component of the VOR by approximately 50%. This result indicates that the gain of the VOR is effectively increased during nitrous oxide induced narcosis. It appears that the vestibular end organs and/or the central pathways controlling nystagmus are affected by nitrous oxide and this may be a reason for the disruption in balance associated with inert gas narcosis.

narcosis
nitrous oxide

vestibular
human

Inert gas narcosis is a behavioral condition that leads to a slowing of response time, sensations of euphoria, loss of memory, and disruptions of balance (1). In previous studies, the effects of narcosis on balance have been examined using measures of total body sway (2, 3). The results have shown that postural stability deteriorates with increases in the partial pressure of nitrogen. It is known that a close relationship exists between postural stability and the vestibular ocular reflex (VOR) (4) and thus narcosis could also affect the VOR.

The VOR is a reflexive type eye movement, which takes place in response to head rotation. Under normal, illuminated conditions the slow tracking phase component of this reflex occurs at a velocity equal to that of the head but in an opposite direction. Each slow phase event is automatically followed by a fast phase epoch, which returns the eyes to their original position. This process is called nystagmus (4). The purpose of the VOR is to maintain the stability of the eyes in inertial space during head motion. The present study was undertaken to determine whether the VOR is disrupted during narcosis induced by nitrous oxide.

METHOD

The VOR was examined using a repeated measures experimental design based on 6 informed, consenting male subjects ranging in age from 25 to 31 yr. The subjects were tested under three conditions, each on successive days. In conditions one and three, the subjects breathed a standard air mixture as a control. In condition two, the subjects breathed a mixture containing 25% nitrous oxide and 75% oxygen. All breathing mixtures were administered through a nonrebreathing oronasal mask (Canadian Forces, model A13A).

The VOR was assessed using a torsion swing (specially built inhouse for this purpose) which turned in a horizontal plane, through an angle of 180 degrees. The swing was spring-loaded and produced a series of decaying oscillations. The frequency of the torsion swing was set at about 0.2 Hz and the oscillations continued for approximately 2 min. A potentiometer mounted on the axis of rotation enabled the swing position to be recorded continuously. The subjects were fitted with black-out goggles and sat in the swing with their eyes closed to exclude visual stimulation. In addition, the head was firmly secured by means of a head brace.

Conjugate horizontal eye movements were recorded using electronystagmography (ENG), with silver-silver chloride electrodes (Beckman, E5SH) attached to the outer canthus of each eye and a ground electrode placed on an ear lobe. Eye movements were amplified through an isolated preamplifier (Gould, model P-N11-5407-58) and a universal amplifier (Gould, model 11-4143-01). The electrical signals were recorded by means of a strip chart polygraph (Gould, model 2400S) at a paper speed of 25 mm/s.

The parameter of nystagmus examined was the slope of the slow phase component of the ENG. This parameter, which represents the velocity of the eye tracking phase, was measured and compared to the stimulus or swing velocity for air and nitrous oxide, as illustrated in Fig. 1. The measures were then used to determine gain in the linear system sense. The gain of the VOR was calculated by determining the velocity of the slow phase component of the nystagmus (V_{sp}) relative to the stimulus velocity (V_{st}) $\text{Gain} = V_{sp}/V_{st}$. Mean gain was calculated for each subject based on 10 samples for each air and nitrous oxide condition.

Data for the two air controls and the nitrous oxide condition were analyzed using a repeated measures analysis of variance followed by a Student-Newman-Keuls post hoc test. The level of statistical significance was set at the 0.05 level.

RESULTS

Mean gain as a function of breathing mixture (summed across subjects) is shown in Table 1. A significant ($F = 19.53$, $df = 2,8$, $P < 0.01$) difference was found between the three conditions. This difference resulted from an increase of about 50% ($P < 0.01$) in the gain of the VOR during the nitrous oxide condition, as compared to either of the controls. There was no difference between the two air control conditions.

DISCUSSION

The results indicate that nitrous oxide affects the gain of the VOR by increasing the slow phase velocity of the nystagmus. This can be interpreted as an increase in

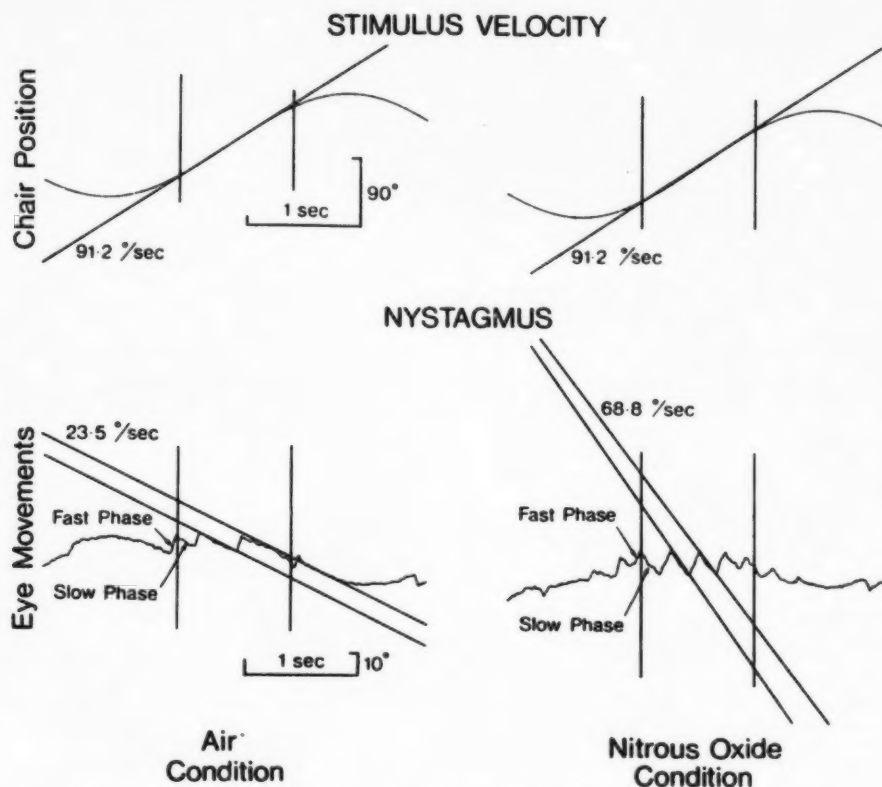


Fig. 1. An example of the data recorded from a single subject illustrating the swing and nystagmus (eyes closed) signals under air and nitrous oxide.

TABLE 1
MEAN GAIN ($\pm$ SEM) OF THE VESTIBULAR OCULAR REFLEX AS A FUNCTION OF BREATHING MIXTURE

Air Control before Nitrous Oxide	Breathing Conditions	
	Nitrous Oxide	Air Control After Nitrous Oxide
0.41 $\pm$ 0.11	0.62 $\pm$ 0.11	0.42 $\pm$ 0.09

the velocity of the tracking component. A similar effect has been shown at very high pressure but the cause remains unclear (5). Regardless, the finding that narcosis affects the VOR is in conflict with a previous study by Adolfson et al. (6) who found no effect with hyperbaric air. One possible reason for this difference is that these researchers used hyperbaric air to induce narcosis instead of nitrous oxide. However, this is an unlikely explanation because there is substantive evidence that nitrous oxide and hyperbaric air have qualitatively identical effects on performance (1).

A more likely explanation for the discrepancy is that the two studies measured different aspects of the nystagmus. Adolfson et al. (6) measured the number of beats and the phase difference between stimulus and response, whereas the present study measured the velocity of the slow phase component relative to the stimulus velocity. It may be that the velocity of the slow phase component is a more sensitive measure of narcosis.

It remains to be confirmed that hyperbaric air produces the same effect on the slow phase velocity of the VOR as nitrous oxide. However, the fact that both hyperbaric air and nitrous oxide influence body sway in the same manner (7) suggests that the effects on VOR and body sway are manifestations of balance disruption due to narcosis.

Measures of total body sway reflect the dynamic interplay of a number of sensory systems. These include somatosensory pressure sensors on the base of the feet, proprioceptive receptors in skeletal tendons and joints, as well as visual, auditory, and vestibular pathways (4). On the other hand, the VOR reflects the integrity of the vestibular end-organs and CNS pathways which control nystagmus. The increase in the VOR gain indicates that the disruption of postural stability during narcosis is probably due, in part, to the impairment of either or both of these systems.

Additional studies are needed to examine the slow phase component of the VOR during exposure to hyperbaric air to confirm that nitrous oxide and hyperbaric air produce identical effects.

The authors thank Barry Kelso and Masao Ohmi for their technical support throughout this project.
DCIEM research paper no. 86-P-36.

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Hamilton K, Porlier G, Landolt J, Fraser W, Fowler B. Effets de la narcose des gaz inertes sur le réflexe oculaire vestibulaire. *Undersea Biomed Res* 1986; 13(3):355-359.—Une étude fut effectuée afin d'examiner le réflexe oculaire vestibulaire (VOR) durant la narcose. La vitesse de la phase lente du nystagmus fut mesurée chez 6 sujets à l'aide de l'électronystagmographie durant l'inhalation de 25% de protoxide d'azote. Il a été découvert que le protoxide d'azote augmenta la vitesse de la phase composante lente du VOR par approximativement 50%. Ce résultat indique que le gain du VOR est effectivement augmenté durant la narcose induite par le protoxide d'azote. Il apparaît que la terminaison des nerfs afférents vestibulaires et/ou les voies centrales, qui contrôlent le nystagmus, sont affectées par le protoxide d'azote et ceci peut être une raison pour le déséquilibre associé avec la narcose des gaz inertes.

narcose
protoxide d'azote

vestibulaire
humain

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